SNS COLLEGE OF ALLIED HEALTH SCIENCE

Affiliated to The Tamil Nadu Dr MGR Medical University, Chennai

DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE TECHNOLOGY

COURSE NAME : PATHOLOGY

TOPIC : CELLS INVOLVED IN INFLAMMATION

UNIT : 2

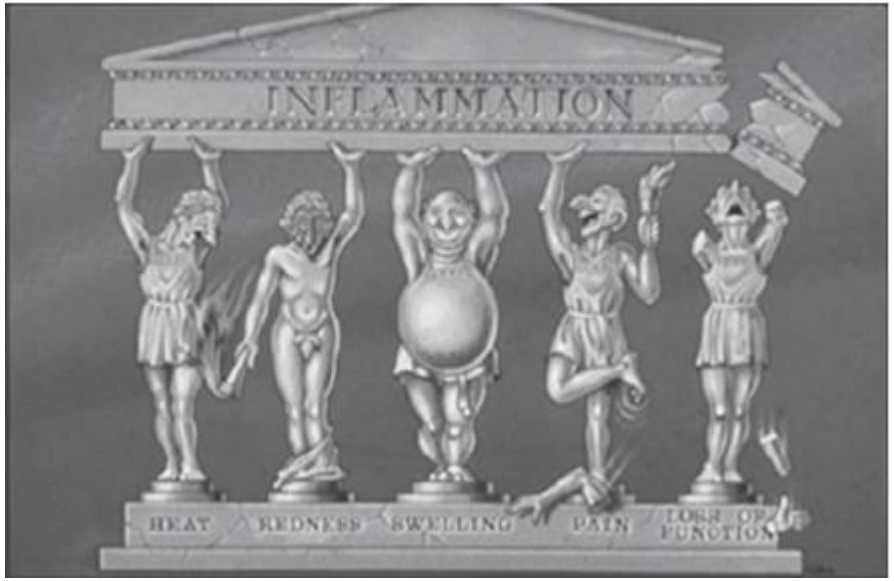
FACULTY NAME : Ms. NIVETHA RAJA

INTRODUCTION

DEFINITION { DEFINE STAGE } :

A VITAL DEFENSE

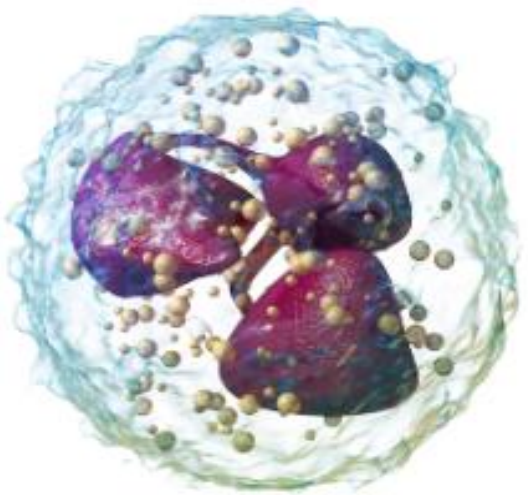
Inflammation is a fundamental protective response by the body's immune system to harmful stimuli, such as pathogens, damaged cells, or irritants. Its goal is to eliminate the initial cause of cell injury, clear out necrotic cells, and initiate tissue repair.



THE FIRST RESPONDERS : NEUTROPHILS

THE VANGUARD OF DEFENSE

- Neutrophils are the most abundant white blood cells and the hallmark of acute inflammation. They are the first to arrive at the scene of injury or infection.
- **PHAGOCYTOSIS:** Engulf and destroy bacteria and fungi.
- **DEGRANULATION :** Release powerful enzymes (like myeloperoxidase) to kill pathogens.
- **NETs:** Form Neutrophil Extracellular Traps (NETs) to capture microbes.



THE CLEAN UP CREW : MACROPHAGES

THE SITE MANAGERS

Derived from **monocytes,** macrophages are versatile cells crucial in both **acute and chronic inflammation**. They manage the inflammatory site.

PHAGOCYTOSIS: Clear pathogens, dead cells (including old neutrophils), and debris.

CYTOKINE RELEASE : Secrete signaling molecules (like TNF-α, IL-1) to coordinate the immune response.

TISSUE REPAIR : Transition to an anti-inflammatory state to promote healing

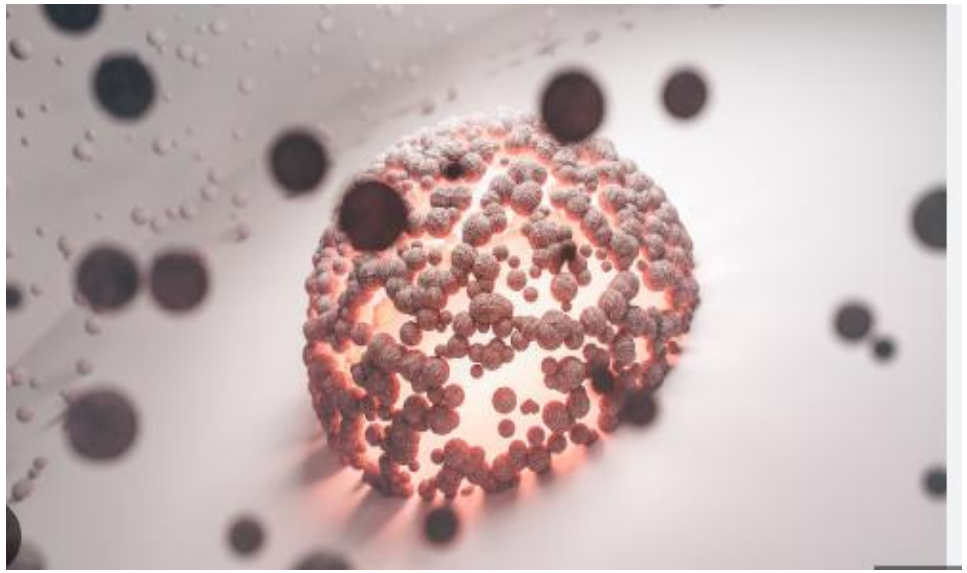
THE ENFORCERS : THE GIANT CELLS

MACROPHAGE SPECIALIZATION :

Formed by the fusion of macrophages, these are the '**heavy-duty**' clean-up crew. They are a hallmark of granulomatous inflammation (e.g., in Tuberculosis).

WALL OF THREATS : Their main job is to contain persistent pathogens or foreign bodies.

LARGE SCALE DEFENSE : They surround material that is too large for a single macrophage to clear.



THE ALARM SYSTEM: THE MAST CELLS

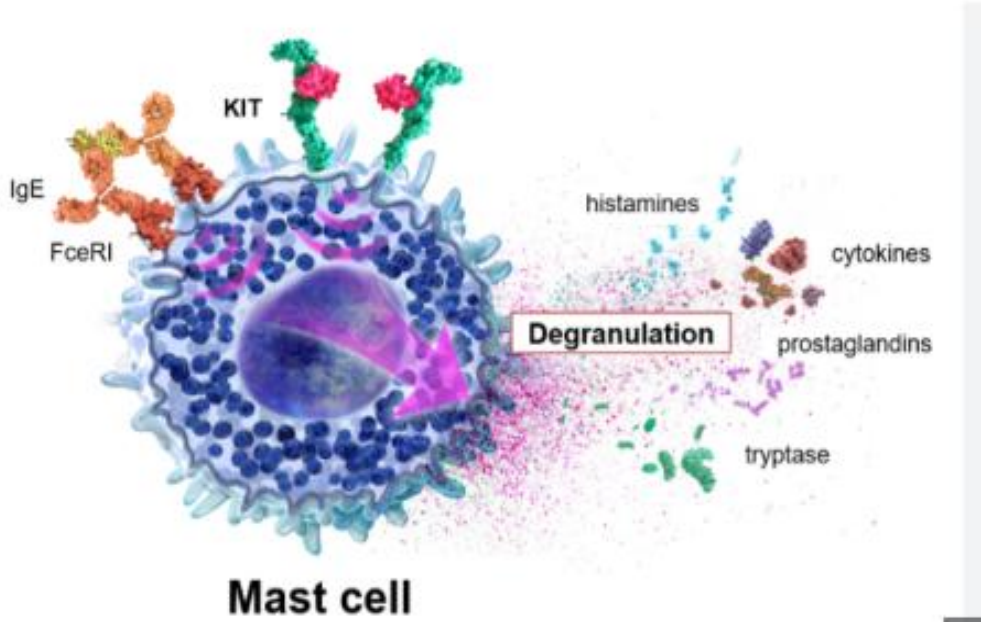
THE SENTINELS

Stationed in tissues (like skin and mucosa), mast cells are sentinels that sound the alarm. They are key in allergic reactions and the initial vascular response.

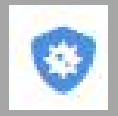
DEGRANULATION: Rapidly release granules containing histamine.

VASODILATION : Histamine causes blood vessels to widen, increasing blood flow (redness, heat).

PERMEABILITY: Increase vascular permeability, allowing plasma and cells to enter the tissue (swelling).



THE CHRONIC INFLAMMATION SPECIALISTS



LYMPHOCYTES :

T-cells and B-cells are the main drivers of chronic inflammation and adaptive immunity. They provide a specific response and create immune memory. B-cells are the precursors to Plasma Cells

EOSINOPHILS :

Primarily involved in allergic reactions and defense against parasites. They release toxic proteins to kill larger invaders that are too big for phagocytosis.

BASOPHILS :

Similar to mast cells, they circulate in the blood and release histamine and heparin, contributing to allergic responses and modulating inflammation.

THE FACTORIES : PLASMA CELLS

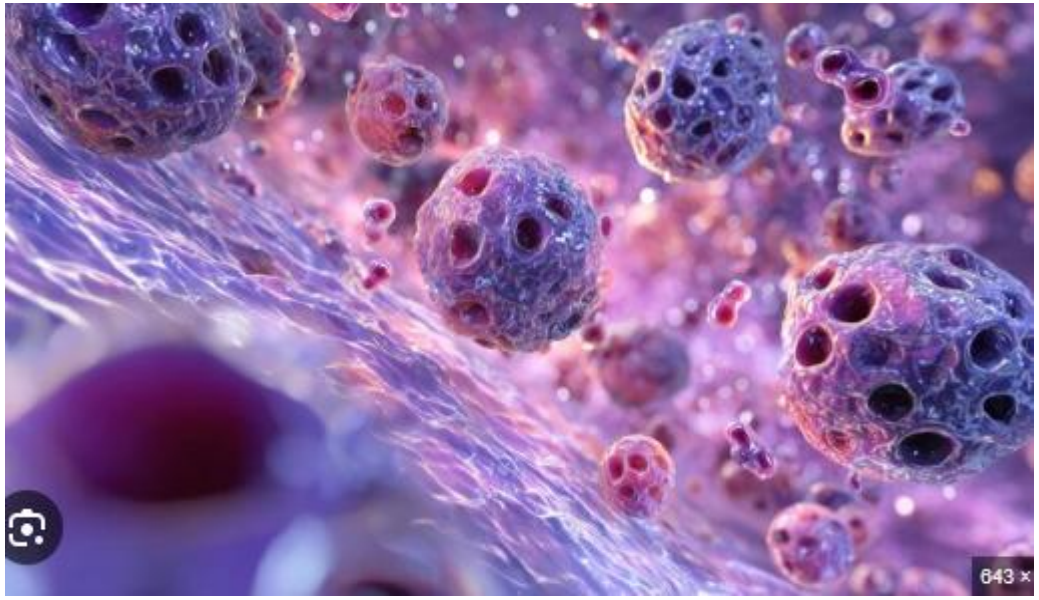
ANTIBODY PRODUCTION :

Plasma cells are fully **differentiated B-lymphocytes.** They are **"antibody factories"** that produce massive amounts of specific antibodies to neutralize persistent antigens.

B-CELL MATURATION : Represent the final stage of B-cell lineage.

TARGETED DEFENSE : The antibodies they produce tag specific pathogens for destruction.

CHRONIC HALLMARK : Their presence is a key feature of established chronic inflammation.

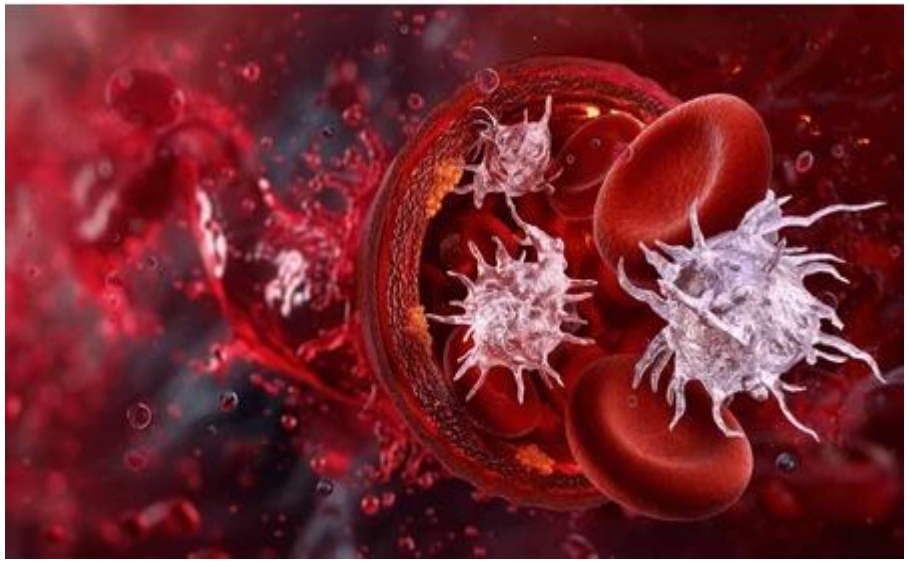


THE “OTHER” CONTRIBUTORS

MORE THAN THEIR JOB

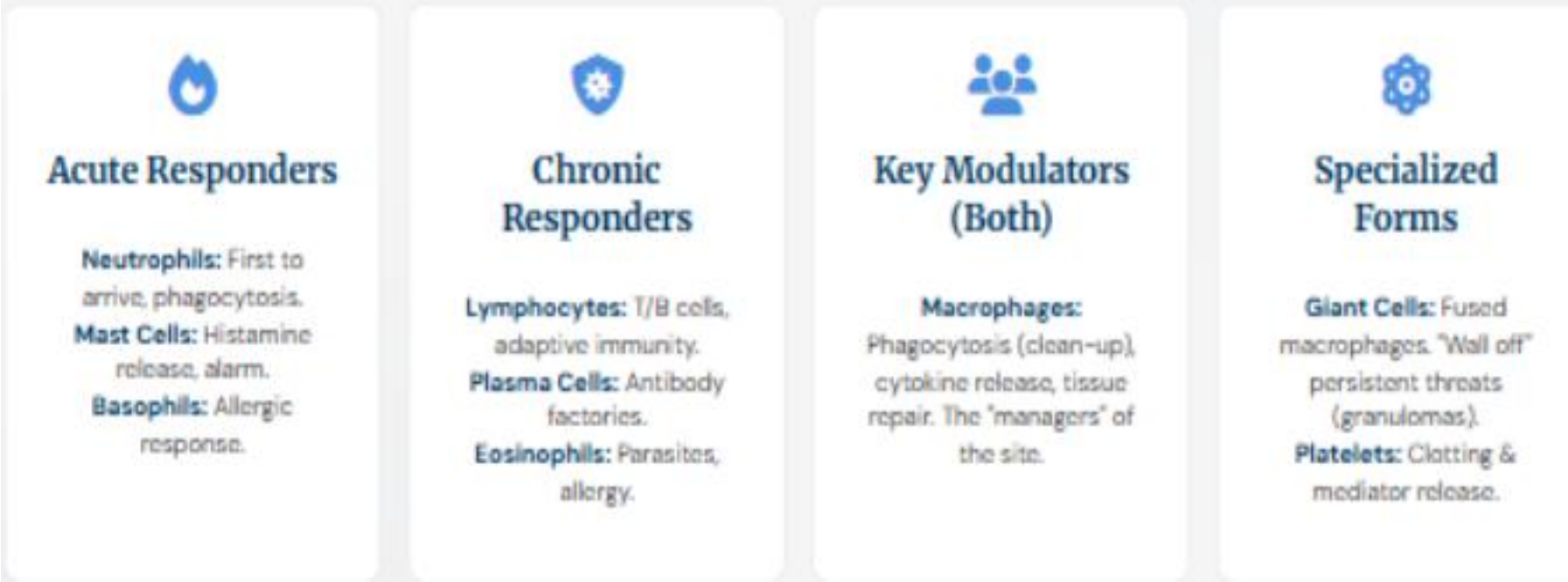
- **PLATELETS (THROMBOCYTES):** Beyond clotting, platelets are active in inflammation. They release mediators that attract other immune cells and promote vascular changes.

- **ENDOTHELIAL CELLS :** The lining of blood vessels isn't just a barrier. These cells actively control inflammation by expressing adhesion molecules, allowing neutrophils to 'stick' and exit the bloodstream into the tissue.



SUMMARY

VISUAL MINDMAP



ACUTE VS CHRONIC CELLULAR RESPONSE

FEATURE	ACUTE INFLAMMATION	CHRONIC INFLAMMATION
Onset	Rapid (minutes to hours)	Slow (days to weeks)
Primary Cells	Neutrophils, Macrophages	Lymphocytes, Plasma Cells, Macrophages, Giant Cells
Key Mediators	Histamine, Prostaglandins	Interferon-gamma, Cytokines
Outcome	Resolution, healing, or abscess	Tissue destruction, fibrosis, scarring

REFERENCES

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